REMARKS

Favorable reconsideration of the subject application is respectfully requested in view of the following comments.

Claims 1-5, 7, 9, 11 and 13-20 are pending in the present application.

I. Rejection of Claims 5, 7, 9, 11, and 13-20 Under 35 U.S.C. § 103(a) Over Wada *et al.*

Claims 5, 7, 9, 11 and 13-20 are rejected under 35 U.S.C. § 103(a) as being unpatentably obvious over Wada *et al.* The Examiner states that Wada teaches use of a compound encompassed by the generic formula of claim 5 and further discloses that the compound is useful in the treatment of seizures associated with epilepsy. The Examiner concludes, therefore, that it would have been obvious to one of ordinary skill in the art to modify Wada's teachings of research results with an experimental rat model of seizures to use with humans.

Applicants respectfully disagree with the Examiner's conclusion.

Wada teaches that administration of the compound, CPBG, to rats "increases the duration of fully kindled seizures and facilitates the developmental seizure process. . .." In other words, administration of CPBG to rats **causes seizures** in this animal model, and this reference cannot be read as suggesting the use of agmatine or its analogs in the treatment of human epilepsy. Contrary to the Examiner's assertions this reference teaches away from the claimed invention.

Accordingly, the rejection of claims 5, 7, 9, 11 and 13-20 are rejected under 35 U.S.C. § 103(a) as being unpatentably obvious over Wada *et al.* is respectfully traversed.

II. **Rejection of Claims 5, 7, 9, 11, and 13-20 Under 35 U.S.C. § 103(a) Over Uzbay *et al.* and Rajasekaran**

Claims 5, , 7, 9, 11, and 13-20 are rejected under 35 U.S.C. § 103(a) over Uzbay *et al.* and Rajasekaran. The Examiner states that Uzbay teaches use of 40 mg to effectively treat audiogenic seizures associated with alcohol withdrawal (in rats). The Examiner also relies on Uzbay as teaching that the therapeutic effects in rats is due to blocking NOS and the NMDA subclass of glutamate receptor channels. Rajasekaran teaches anticonvulsant activity of agmatine in the treatment of seizures due to epilepsy and that the underlying mechanism for the anticonvulsant activity is NO inhibition. The Examiner concludestherefore, that it would have been obvious to modify Uzbay's teaching in view of Rajasekaran to treat seizures due to epilepsy.

Applicant respectfully disagrees with the Examiner's conclusion.

The teachings of Uzbay *et al.* are clearly limited to and relevant only to seizures associated with alcohol withdrawal. In particular, this reference teaches that agmatine is useful in the treatment of audiogenic seizures associated with ethanol withdrawal. The authors concludes that agmatine "has some inhibitory effects on the withdrawal syndrome in ethanol-dependent rats." The authors suggested that three possible mechanisms might be responsible for the observed inhibitory effects on ethanol withdrawal, but did not draw any conclusions concerning the mechanism of action of agmatine on inhibition of seizures and other symptoms associated with ethanol withdrawal, and clearly did not suggest the use of agmatine or its mechanism of action on any other types of seizures.

The secondary reference, Rajasekaran *et al.*, teaches that NO may be an important pathogenic component in the mechanisms that regulate seizure induction, but the "role of NO in epilepsy remains debatable." The conclusions drawn by the authors from their experiments on

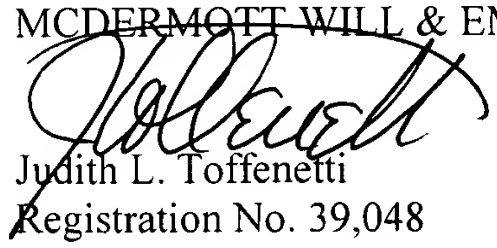
pretreatment of rats with arginine prior to induction of experimentally induced seizures is that inhibition of NO may facilitate seizures, but the abstract does not offer any evidence of a link between seizure control, NO inhibition and agmatine. Thus, the combined prior art fails to render the claimed invention obvious.

It is respectfully submitted that the present application is in condition for allowance, an early notification thereof being earnestly solicited.

To the extent necessary a petition for an extension of time under 37 C.F.R. § 1.136 is hereby made. Please charge any shortage in fees in connection with the filing of this paper, including extension of time fees, to Deposit Account 500417 and please credit any excess fees to such deposit account.

Respectfully submitted,

MCDERMOTT WILL & EMERY LLP



Judith L. Toffenetti
Registration No. 39,048

600 13th Street, N.W.
Washington, DC 20005-3096
202.756.8000 JLT:ajb
Facsimile: 202.756.8087
Date: September 8, 2004